LOWER WILLAMETTE GROUP

Chairperson: Bob Wyatt, NW Natural
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February 6, 2009

Chip Humphrey
Eric Blischke
U.S. Environmental Protection Agency, Region 10
805 SW Broadway, Suite 500
Portland, OR 97205

Re: Fish Tissue Residue Toxicity Reference Values for the Baseline Ecological Risk Assessment (Lower Willamette River, Portland Harbor Superfund Site, USEPA Docket No: CERCLA-10-2001-0240)

Chip and Eric:

Thank you for your January 23, 2009 letter, in which you directed the Lower Willamette Group (LWG) to include the 11 specific lowest observed effects residues (LOERs) in the development of fish tissue residue TRVs for use in the Portland Harbor BERA.

The LWG does not accept your assertions that EPA has objectively evaluated our technical arguments, and determined that inclusion of the 11 LOERs is necessary to ensure that the Portland Harbor BERA is conducted appropriately and in a manner consistent with the previously agreed upon TRV methodology. Specifically, we reject your arguments for including six of the 11 LOERs. We have communicated the specific rationale for rejecting the studies based on technical reasons having to do with the facts of the individual studies, and or conflicts with the process that the EPA previously approved for developing TRVs.

Because EPA has directed the LWG to include the values, the LWG will include the 11 LOERs in the development of fish tissue residue TRVs as required by Section IX.1 of the Administrative Settlement Agreement and Order on Consent.

Following is a summary of our objections to the aforementioned assertions.

Behavioral Endpoints: Following review of the Fish Tissue Residue Toxicity Reference Value (TRV) Reconciliation Tables submitted by the LWG to EPA on November 20, 2008, EPA identified exclusion of behavior studies as a primary area of disagreement with LWG regarding exclusion of LOERs from the species sensitivity distributions used to derive fish tissue-residue TRVs. EPA's memo dated December 22, 2008 provided evidence linking prey capture ability, avoidance behavior, feeding behavior, and swimming activity to adverse effects at the population or community level. On January 14, 2009 the LWG provided you with its evaluation of the degree to which the evidence EPA provided for population level adverse effects described for these behaviors can be extrapolated to other studies:

1. Predator-prey relationships

   EPA used the example of Weiss et al. (2000) to support inclusion of studies reporting predator-prey relationships in SSDs. Weiss et al. (2000) provide literature as well as field and laboratory experimental evidence showing causal linkages between mummichog exposure to chemical contaminants (primarily mercury), reduced prey capture ability, and reduced growth and lifespan. The specific behavioral endpoint reported was time required to capture prey. A possible causal mechanism that was experimentally described was increased brain mercury concentrations causing decreased neurotransmitter levels. Linkages between organism and population level traits were demonstrated for a single contaminated site where several contaminants were present. The LWG found that study convincingly tied reduced mummichog growth and life-span at the contaminated site investigated to decreased ability to capture prey, but that applying these results to predator-prey studies in general constitutes extrapolating outside the dataset. Nonetheless, we concluded that from a conservative perspective this study supports inclusion of studies reporting reduced prey capture rate in SSDs, and that more generally it suggests that any endpoint directly related to reduced prey capture ability might influence population level endpoints. Therefore, pursuant to EPA direction, LWG has agreed to include all studies reporting prey capture rate LOERs in SSDs, and to consider studies reporting other predator-prey interactions on a case by case basis.

2. Avoidance behavior

   No avoidance behavior studies were included in the SSD database, therefore evidence supporting inclusion of studies reporting this category of behaviors was not reviewed.

3. Feeding behavior

   EPA used the example of Weiss et al. (2000) (discussed above under predator-prey relationships) to support inclusion of studies reporting feeding behavior in SSDs. Weiss et al. (2000) report that mummichog from a contaminated site fed on detritus, whereas at an uncontaminated reference site they fed on mobile prey. No specific feeding behaviors other than prey capture rate (described above) were investigated. EPA also referenced Buckley et al. (1982) as evidence that reduced feeding by coho salmon resulted in reductions in growth. Buckley et al. (1982) reports that feeding rates were initially depressed in copper exposed fish relative to controls but recovered to control levels with time and that weight of exposed fish (except at the highest exposure level) also became similar to that of controls near the end of the 100 day aqueous copper exposure. At the highest treatment level, the observed growth effect was potentially attributable to loss of food from the tank due to reduced movement of the fish. This study demonstrates that the effect of reduced feeding rate on population level endpoints is linked to the persistence and magnitude of the reduced feeding rate effect. Therefore, the LWG agreed to evaluate studies reporting feeding behaviors on a case-by-case basis to determine whether the magnitude and duration of the effect is likely to result in reduced growth, and if so to use that study in fish tissue TRV derivation.

4. Swimming activity

   EPA used the example of Smith and Weis (1997) to support inclusion of studies reporting effects on swimming activity in SSDs. Smith and Weis (1997) did not experimentally investigate any swimming behaviors. The nearest endpoints evaluated were the number of strikes by mummichog on grass shrimp made per fish per minute, the number of strikes per fish per kill, and the number of strikes per pursuit. The authors found that mummichog from a contaminated site had fewer strikes on grass shrimp than mummichog from a reference site. They suggest that a possible mechanism for this effect is related to contaminant-related changes in neurotransmitter levels causing reduced swimming activity. The evidence presented by EPA thus did not demonstrate causal relationship between swimming activity and population-level adverse effects. Therefore, the LWG concluded that it was necessary to reevaluate swimming behavior studies on a case-by-case basis.

LWG's conclusions regarding the general evidence for inclusion of these types of behavioral endpoints indicated the need to reevaluate the specific papers in question on a case-by-case basis. LWG and EPA collectively discussed the papers and the LWG's conclusions during the January 9 meeting. The result of that discussion was EPA requesting a summary of the LWG's analyses, clearly stating which specific behavioral studies were rejected based on grounds other than use of a behavioral endpoint. The LWG submitted that work to EPA on January 14. Twenty of the 27 LOERs were rejected on grounds other than use of a behavioral endpoint. Of the remaining seven, the LWG rejected two and accepted five. The two that were rejected are Davy et al. (1972) and Webber & Haines (2003). The LWG accepted Weber et al. (1991), Kania & O'Hara (1974), Begtsson (1980) and two LOERs from Gakstatter & Weiss (1967).

In your January 23 letter you directed the LWG to accept six of the seven behavioral LOERs that weren't rejected for some other reason besides the use of the behavioral endpoint, i.e., the five that the LWG had already agreed to include plus Webber & Haines (2003). The LWG's technical position continues to be that Webber & Haines (2003) should be rejected for tissue TRV derivation based on established acceptability criteria.

- Webber & Haines (2003) reported that golden shiner exposed to mercury "had significantly greater shoal vertical dispersal following predator exposure, took longer to return to pre-exposure activity level, and had greater shoal area after return to pre-exposure activity than controls." Although the authors contended that these behaviors would increase vulnerability to predation, they did not present evidence linking these behaviors to population level effects to support their contention. Therefore the LWG rejected this study for use in fish tissue TRV derivation because a direct link to reduced survival, growth or reproduction has not been established.

Of the remaining 20 behavioral studies that the LWG rejected for reasons other than the behavioral endpoint, EPA has agreed to reject 17 and directed the LWG to use three. Those three studies are: Hollis et al. (2000), Fisher et al. (1994) and Scott et al. (2003). The LWG's technical position continues to be that all three of these studies should be rejected for tissue TRV derivation based on established acceptability criteria.

- Hollis et al. (2000) reported a LOER for reduced survival and impaired swimming. However, the authors report that "(m)ortality was minimal for all treatments (up to 14% for 0.11 µg/l Cd). No significant effects of chronic Cd exposure were seen in growth rate, swimming performance (stamina), routine O_2 consumption, or whole body/plasma ion levels." Therefore, the LWG rejected Hollis et al. (2000) for fish tissue TRV derivation because no adverse effects were associated with chronic Cd exposure.

- Fisher et al. (1994) is a PCB study that EPA apparently had not reviewed as of our January 9 meeting on this issue. The study reports LOERs for reduced growth and retarded phototropism behavior in alevins. Fish in this study were exposed only as eggs; therefore the study failed a study acceptability criterion that the LWG and EPA had agreed to for tissue TRV derivation, and the LWG rejected it.

- Scott et al. (2003) experimentally investigated juvenile rainbow trout response to alarm substance extracted from fish skin. At the highest aqueous treatment level, Cd exposed fish did not stop feeding, did not seek shelter, and continued to move similarly to when the alarm substance was not present whereas controls behaved in the opposite fashion. Behavior of fish exposed through diet, and at lower aqueous treatment levels was not statistically different from controls. The dietary and highest aqueous exposures resulted in similar tissue burdens and the authors suggest that tissue burdens in the olfactory system determine toxicity. Therefore, there is reasonable doubt as to whether the whole body tissue residue accurately represents a true effects threshold, but the LWG acknowledged that similar uncertainty applies to all short term effects studies and to metals in particular. Despite this concession to uncertainty, the LWG found that neither the study itself nor EPA provided evidence directly linking the observed short term predator avoidance effects to reduced survival, growth or reproduction. Therefore the LWG rejected this study for use in tissue TRV derivation.

Inclusion of 1970s Great Lakes Sac-Fry Studies: In January 7 correspondence and at the January 9 fish tissue TRV meeting, the LWG argued that inclusion of the Berlin et al. (1981) and Broyles and Noveck (1979) studies is inconsistent with the LWG/EPA agreed-upon tissue TRV methodology. Our arguments were provided to EPA in January 14 and January 21 correspondence and in a telephone conversation between an LWG representative and Eric Blischke on January 22, 2009. The LWG's technical position continues to be that these two studies should be rejected for tissue TRV derivation based on established acceptability criteria.

- Berlin et al. (1981) showed that fry hatched from eggs from Lake Michigan lake trout (with a measured total PCB egg residue of 7.6 ppm and day-old fry residue of 3.8 ppm) chronically exposed to Aroclor 1254 water concentrations from 1x to 25x ambient concentrations in Great Lakes surface water (circa 1975) exhibited significant excess mortality. The LWG concluded that it is wrong to use the data in Berlin et al. (1981) to calculate a tissue TRV because exposure and effects weren't measured at the same time. Significant excess mortality occurred in days 57-96 and (to a lesser extent) days 97-136, but tissue residue wasn't measured until the end of the 176-day experiment, at which time the tissue residue was lower than at the beginning of the experiment (i.e., the initial tissue concentration due to maternal transfer of PCBs obtained from Great Lakes exposure was higher than the final tissue concentration. Therefore, the LOER based on measurements

taken at the end of the study is an underestimate and should be rejected for tissue TRV development.

Broyles and Noveck (1979) showed that sac-fry hatched from Lake Michigan lake trout and Chinook salmon (with total PCB egg residues estimated to be in the 3-11 ppm range) exposed to low ppb water concentrations of PCB 153 caused excess mortality. The LWG concluded that it is wrong to use of the data in Broyles and Noveck (1979) to calculate a tissue TRV because the study only provides tissue residue data for the ^{14}C-labeled PCB 153 fraction of the total PCB tissue residue. The study did not account for the tissue burden resulting from maternal transfer. Therefore, the LOER based on measurements of ^{14}C-labeled PCB 153 is an underestimate of the tissue residue and should be rejected.

In conclusion, the LWG rejects your assertions regarding the appropriateness of six of the 11 LOERs that you have now directed us to use for tissue TRV development. Our counter-arguments to your assertions have been restated in this letter. Nonetheless, the LWG will comply with EPA's direction to use these LOERs in TRV development for the BERA. The LWG reserves the right to object to specific applications of the TRVs to establish remediation goals for the Portland Harbor site.

It is our understanding that this concludes the process of fish tissue TRV development for the BERA, and specifically that EPA will not be asking or directing the LWG to make any changes to the November 20, 2008 fish tissue residue TRV reconciliation tables beyond what is directed in your January 23 letter.

On a related matter, the LWG has not received direction on the benthic tissue TRVs, so we are planning to proceed with the benthic tissue TRVs we submitted to you on November 14, 2008, with the cadmium, copper and DDD updates requested by Burt Shephard, and submitted to him on November 26, 2008. With the exception of PCBs, we reached verbal agreement on these TRVs, though that has not been formalized. With regard to the PCB TRV, you might recall that you were considering asking us to add the papers by Dillon et al. (1990) and Fowler et al. (1978) to the LOER dataset. The last we heard from you on this matter was that the question was on the agenda for your December 3, 2008 agency team meeting.

It might be useful for you to know that the decision about whether to include these two studies has no effect on the benthic tissue PCB TRV (either way the TRV is 1.2 mg/kg-ww). The LWG finds the LOER from Dillon et al. (1990) to be unacceptable because it's a single congener (PCB 101) study that didn't meet our criteria for inclusion. The LWG finds the LOER from Fowler et al. (1978) to be unacceptable because of weak study design (inadequate documentation of effect). Given that these are marginal LOERs about which we disagree, and that they have no effect on the TRV, the LWG plans to proceed using the November 14, 2008 benthic tissue PCB TRV submittal, unless directed to update and resubmit the benthic tissue TRV reconciliation table for PCBs.

Sincerely,

Bob Wyatt

cc: Confederated Tribes and Bands of the Yakama Nation
Confederated Tribes of the Grand Ronde Community of Oregon
Confederated Tribes of Siletz Indians of Oregon
Confederated Tribes of the Umatilla Indian Reservation
Confederated Tribes of the Warm Springs Reservation of Oregon
Nez Perce Tribe
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